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Articular Surface Defects in the Third Metatarsal and Third Cuneiform: Nonosseous Tarsal Coalition

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ABSTRACT Frequencies of articular surface defects on the third metatarsal and third cuneiform, seen as pits of varying sizes on the plantar one third of the tarsometatarsal articular face, were investigated in skeletal populations from North America and Japan, as well as in gibbon, orangutan, chimpanzee, and gorilla skeletons. The apes did not exhibit the defects, although the number of observed specimens of each type was small. The newly presented human frequencies corresponded well with those from other published sources. The defects appeared both unilaterally and bilaterally, with no apparent sex or side biases. Statistical tests between the various populations found that, in general, geographically close populations had more similar frequencies of the defect. Possible etiologies for the defect were investigated, including biomechanical influences, degenerative arthritis, infection, trauma, and a developmental condition known as tarsal coalition, which proved to be the best explanation. Tarsal coalition results from the failure of a joint space to form properly during fetal growth. It can occur between any two adjacent bones of the foot. Several clinically important coalitions, whose presence interferes with normal walking, are known. However, coalition between the third metatarsal and third cuneiform has not been reported in the clinical literature, suggesting that the defect causes little or no foot dysfunction. Tarsal coalition is thought to have a strong genetic component, suggesting that the pit defect may be useful as a skeletal nonmetric trait, as others have stated. Am J Phys Anthropol 109:53–65, 1999.

Defects involving the common articular surface between the third metatarsal (MT3) and third cuneiform (CF3) have been reported in skeletal samples from both the Old and New Worlds, with frequencies ranging from 3.2–26.0% (Case, 1996; Regan and Case, 1997; Tenney, 1991; Wilbur, 1997). These defects are typically present as a circular or oval pit in the proximal facet of the third metatarsal and the distal facet of the third cuneiform, and are usually restricted to the plantar one third of these facets. Although the defects were previously described as a nonmetric trait (Tenney, 1991), their etiology remained unclear. We were intrigued by the regularity in location and appearance of these defects and felt that further inquiry was warranted. Illustrations of the many varieties of tarsal coalition (Sarrafian, 1993), along with a published description of nonosseous coalition between two other tarsal bones (Hynes and Romash,

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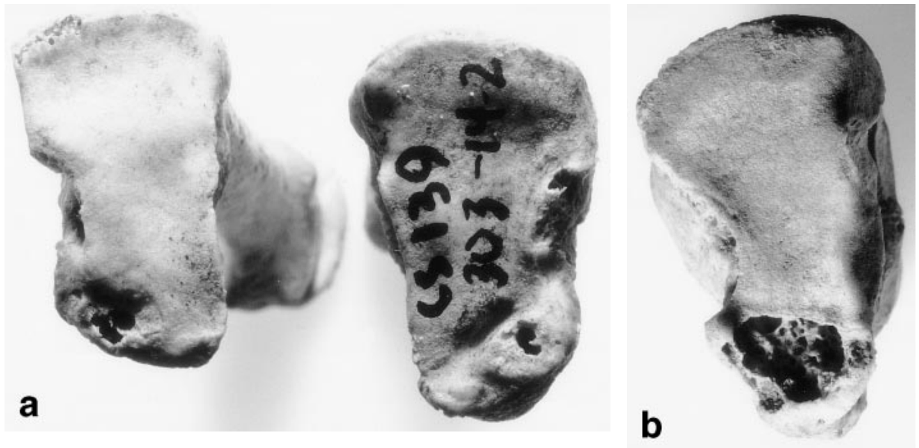

Fig. 1. Range of expression of the MT3-CF3 defect. **a:** Low-grade expression. At right, a crease and small pit on a CF3; at left, a small pit on an MT3. Both bones are from the same foot. **b:** Large pit in a CF3, extending the mediolateral width of the articular face. Photographs by Korri D. Turner.

1987), led us to investigate the possibility that the MT3-CF3 defects represent incomplete or nonosseous tarsal coalition between the two bones. Our differential diagnosis also includes biomechanically induced changes, arthritis, infection, and trauma.

The purpose of this paper is twofold: first, to present new frequency data from several groups and to compare these groups to each other and to previously published frequencies; and second, to investigate the etiology of the MT3-CF3 defect.

DESCRIPTION OF THE DEFECT

The articular surface defects on the proximal third metatarsal and distal third cuneiform are nearly always found on the plantar one third of the facets and are most often seen as round or oval depressions with pitting in the floor of the defect. Expression of the defect seems to vary along a continuum, ranging from a smooth crease or pit the size of a pinhole to a very large depression sometimes several millimeters in depth (Fig. 1a,b). These larger depressions may extend across the mediolateral width of the articular face and exhibit thickened margins that sometimes serve to expand the articular facet beyond its normal limits in the mediolateral and plantar directions. Frequently the plantar tip of the metatarsal or cuneiform will curve toward the opposing bone. When present, the pit defects are always seen on both the third metatarsal and the third cuneiform, with a certain degree of symmetry in their morphology, i.e., small creases or pits on one bone are mirrored by small creases or pits on the other; large pits are similarly reflected across the joint space. This symmetry means that only one of the bones needs to be present in order to score the defect.

MATERIALS AND METHODS

Frequency data were gathered from skeletal remains representing American Indians, nineteenth century Americans, modern Japanese, and Ainu (Table 1). Data from several different species of apes were also gathered to determine if the defect is present outside of humans. Comparative data on other human populations were taken from the published anthropological and anatomical literature (Table 2).

Prehistoric American Indian skeletons include Salado remains from the central Arizona Tonto Basin, dating from circa AD 1150–1450 (Pedrick, 1992; Rice, 1990) and skeletal samples from the Indian Knoll site (15OH2), an archaic site in Kentucky (Webb, 1946). Protohistoric American Indian skeletal data come from the Plains Arikara sites of Mobridge (39WW1) and Sully (39SL4) in

TABLE 1. Frequencies of MT3-CF3 articular surface defects[1]

Sample	n	%	Laterality: Unilateral	Bilateral	Unknown
Mobridge	15/77	19.5	4	6	5
Sully	9/49	18.4	1	5	3
Pima	5/32	15.6	3	2	0
Indian Knoll	55/354	15.5	10	34	11
Salado	17/114	14.9			
Hardin Village	13/103	12.6	3	8	2
Modern Japanese	12/101	11.9	4	8	0
U.S. Civil War	4/48	8.3			
Ainu	0/28	0.0			

[1] Blank spaces indicate data not available.

TABLE 2. Comparative data for MT3-CF3 articular surface defects

Sample	n	%	Source
California Indians (A)	181/695	26.0	Tenney (1991)
California Indians (B)	31/125	24.8	Jurmain (1987)
Prehistoric Illinois	90/496	18.0	Wilbur (1997)
Aleuts/pre-Aleuts	13/99	13.1	Tenney (1991)
Prehistoric New Mexico	5/53	9.4	Lahr (1987)
Terry White	8/110	7.3	Tenney (1991)
Terry Black	9/144	6.3	Tenney (1991)
Predynastic Egyptians	5/121	4.1	Tenney (1991)
German (19th century)	10/313	3.2	Pfitzner (1896)

South Dakota, curated at the Smithsonian Institution and dating from the first half of the eighteenth century (Merchant and Ubelaker, 1977) and from AD 1600–1750 (Owsley and Jantz, 1977), respectively, as well as from the Mississippian-period Hardin Village site (15GP22) in Kentucky, dating from AD 1450–1675 (Hanson, 1966). The two Kentucky samples are curated at the University of Kentucky Museum of Anthropology. Historic American Indian skeletal data (circa AD 1870–1910) come from the south-central Arizona site of Pima Butte (AZ T:16:88 ASM), a historic Pima Indian cemetery excavated as part of a highway mitigation project and since reburied. The modern Japanese data come from late nineteenth and early twentieth century cadavers. The Ainu skeletons were excavated in the latter half of the nineteenth century and are of uncertain date. The Japanese and Ainu collections are curated at the University Museum, University of Tokyo, Japan. Skeletons from the American Civil War are curated at the Armed Forces Institute of Pathology, National Museum of Health and Medicine, Washington DC. Nonhuman primate data were gathered from collections at the Smithsonian Institution in Washington, DC, and include gibbons (n = 14), orangutans (n = 32), chimpanzees and bonobos (n = 19), and gorillas (n = 18).

Comparative data are available from the study by Tenney (1991) of prehistoric California natives, Aleuts/pre-Aleuts, modern American Blacks and Whites from the Terry Collection, and predynastic Egyptians. Frequencies are also known for skeletons from Illinois dating between AD 1–1100 (Wilbur, 1997) and from Germans dating to the late nineteenth century (Pfitzner, 1896).

For this study, defects were scored as either present or absent. Any skeleton with at least one observable MT3 or CF3 was scored for presence or absence of the defect, and frequencies were determined based on numbers of affected individuals. Only skeletons with fused epiphyses were scored for this study because the ability to see this defect in dry bone appears be dependent upon complete development of the relevant articular facets. In practice, this results in a lower age limit of around 14–16 years (Sarrafian, 1993), although we did find one pit defect in an individual aged 12.5–13.5 years from the Schoolhouse Point Mound site in central Arizona (Regan et al., 1996). In addition, Wilbur (1997) mentions a possible pit defect in the left MT3 from a 6-year-old child. Tenney (1991) found no defects in individuals below the ages 9 or 10.

Frequencies in the various samples were compared using a two-tailed Fisher's exact test. In each case, the null hypothesis (H_0) is that the two samples have the same frequency, and the alternative hypothesis (H_a) is that the frequencies are different. Significance was tested at an alpha level of 0.05.

Fig. 2. Chimpanzee MT2-MT5 (right to left), showing small pit defect on proximal MT3 articular facet. The location of this defect is unlike that seen in humans. Importantly, there was no corresponding pit in the articulating CF3. Photograph by Parveen Hamazi.

FREQUENCY DATA RESULTS

Frequencies of the MT3-CF3 pit defect for the study populations are summarized in Table 1. Frequencies reported by other authors are summarized in Table 2. The defect seems to be remarkably common in archaeological and anatomical specimens. Published frequencies range from 3.2–26%. The frequencies for North American Indians are remarkably consistent; most cluster between 12–20%, with a range of 9.4% (Lahr, 1987) to 26% (Tenney, 1991). New frequencies for North American Indians presented herein range from 12.6% for prehistoric Hardin Village Indians to 19.5% for protohistoric Indians from the Sully site in South Dakota. Data from non-North American Indian skeletons indicate frequencies ranging from 0% in native Japanese Ainu to 11.9% in modern Japanese.

Only one ape skeleton showed a pit on an MT3 or CF3. The pit was found in the third metatarsal of a chimpanzee but does not conform to the pattern seen in humans (Fig. 2). The pit is located too high on the facet and, more importantly, is not mirrored in the associated third cuneiform. The lack of pit defects in apes is intriguing, but the small numbers of each species available for observation preclude any evolutionary considerations. More data need to be gathered on apes and other nonhuman primates before any strong conclusions can be reached.

TABLE 3. Fisher's exact test for side biases

Sample	Left	Right	Alpha level	*P*
Arikara[1]	19/111	20/105	0.05	0.73
Japanese	12/97	8/96	0.05	0.48
German[2]	6/268	9/252	0.05	0.44

[1]Contains Mobridge and Sully sites.
[2]From Pfitzner (1896).

Table 1 gives laterality data where they could be determined. Unilateral defects are those that were present on the right or left foot but not on the opposite. Laterality could not always be determined due to incomplete or missing elements. In general, bilateral defects occurred more frequently than unilateral defects. Unilateral defects show no side bias; Fisher's exact tests for left vs. right in three of the study populations from Table 1 returned insignificant *P* values (Table 3). Tenney (1991) and Wilbur (1997) both found that approximately 29% of affected individuals in their samples had unilateral defects, although neither reported if the rest were bilateral or if laterality could not be determined. Wilbur (1997) found no side bias in her data; Tenney (1991) did not statistically test his data.

We similarly investigated possible associations with sex, but found that the occurrence of the defect is independent of the sex of the individual. For example, in our largest sexed series (Indian Knoll), 33/199 males and 22/155 females were affected. However, the frequency difference between the sexes was not significant (alpha = 0.05; P = 0.56).

Tenney (1991) found that affected males outnumbered affected females by a 2.2:1 ratio, but he noted that males were overall more common in his study population. He did not test his sex ratio for statistical significance. Wilbur (1997) found that the pit defect was significantly more common in females than in males; 24% of females in her sample exhibited the defect compared to only 14% of males. The nature of our sample did not allow us to investigate adult age associations, but Wilbur (1997) found no association between adult age and defect occurrence in her large sample from the Midwestern United States.

The combined data from Tables 1 and 2 indicate that North American Indian populations have the highest demonstrated frequencies of the defect. Much lower frequencies are exhibited by German, European-derived, and Egyptian populations. These frequency differences are generally supported by the statistical analyses (Table 4), which suggest that American Indians from California exhibit the highest frequencies; other American Indians and the modern Japanese exhibit moderately high frequencies that do not differ significantly from one another; and the Ainu, Egyptians, Germans, and Terry Blacks generally exhibit low frequencies that are not significantly different from one another. In the few cases in which samples did not follow this trend (e.g., the Pima Indians (15.6% affected) and the Ainu, Terry White, and Terry Black samples), at least one of the two samples being compared tended to be very small (n = 53 or less).

In order to overcome the problem with small samples, we also combined several of the samples based on similarities in ancestry to determine whether the same trends noted in the individual sample comparisons would be supported. The results again show that the combined California Indian sample has a significantly higher frequency of the defect than all other American Indians combined (alpha = 0.05, $P < 0.001$), and that the combined sample of American Indians, excluding the California Indians, has a significantly higher frequency than the combined Terry White, German, and U.S. Civil War sample (alpha = 0.05, $P < 0.001$).

TABLE 4. Pairwise statistical comparisons, using Fisher's exact test, of pit defect frequencies among samples used in this study

Samples	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. California A																	
2. California B	0.825																
3. Mobridge	0.269	0.490															
4. Sully	0.173	0.312	0.814														
5. Prehistoric Illinois	0.002	0.101	0.753	0.847													
6. Indian Knoll	0.000	0.029	0.396	0.836	0.355												
7. Hardin Village	0.003	0.028	0.220	0.616	0.198	0.531											
8. Pima	0.219	0.349	0.788	1.000	1.000	1.000	0.766										
9. Salado	0.010	0.075	0.434	0.816	0.495	1.000	0.696	1.000									
10. Prehistoric New Mexico	0.005	0.024	0.143	0.378	0.128	0.302	0.610	0.492	0.462								
11. Aleuts/pre-Aleuts	0.004	0.041	0.301	0.622	0.248	0.635	1.000	0.770	0.844	0.604							
12. Ainu	0.000	0.001	0.010	0.045	0.008	0.021	0.069	0.055	0.025	0.158	0.070						
13. Modern Japanese	0.002	0.017	0.206	0.453	0.147	0.428	1.000	0.555	0.554	0.789	0.833	0.068					
14. Terry White	0.000	0.000	0.022	0.092	0.004	0.026	0.251	0.168	0.089	0.759	0.174	0.359	0.347				
15. Germans	0.000	0.000	0.000	0.001	0.000	0.000	0.001	0.008	0.000	0.050	0.001	1.000	0.002	0.095			
16. U.S. Civil War	0.005	0.019	0.125	0.356	0.108	0.275	0.584	0.472	0.314	1.000	0.583	0.290	0.585	0.756	0.100		
17. Egyptians	0.000	0.000	0.001	0.011	0.000	0.001	0.026	0.034	0.006	0.175	0.024	0.584	0.041	0.394	0.573	0.275	
18. Terry Black	0.000	0.000	0.005	0.042	0.000	0.005	0.112	0.139	0.035	0.531	0.073	0.358	0.164	0.803	0.137	0.740	0.584

DIFFERENTIAL DIAGNOSIS AND ETIOLOGY OF THE DEFECT

A search of the clinical and anthropological literature turned up only two articles presenting an explanation for the MT3-CF3 defects. Tenney (1991) thought that they were nonmetric traits but did not hypothesize a cause. Wilbur (1997) followed the suggestion of Tenney (1991) and called them a discrete genetic trait, which she believes may be caused by an anomalous interosseous ligament. Our own search for the cause of this defect considered several possibilities: biomechanically induced changes, degenerative arthritis, bone infection, trauma, and tarsal coalition. These etiologies will be considered in turn.

Biomechanics of the third tarsometatarsal joint

The joint between MT3 and CF3 is located at the apex of the transverse arch defined by the tarsometatarsal joints (de Palma et al., 1997). The long axis of the foot passes near this joint (Norkin and Levangie, 1983). Therefore, it is reasonable to consider that biomechanical forces involved in bipedal locomotion might play a role in the appearance of the MT3-CF3 defect. Because the pit defect occurs *only* at the third tarsometatarsal (TMT) joint, we investigated the biomechanical forces of the TMT joints in order to determine if the third TMT joint was affected by forces uncommon to the others.

The MT3-CF3 joint is one of five tarsometatarsal plane synovial joints that join the midfoot to the forefoot. Collectively, these joints are known as the Lisfranc joint, named after a surgeon in Napoleon's army (Myerson, 1989). They act as a continuation of the transverse tarsal joint, which is a compound joint made up of the talonavicular and calcaneocuboid articulations (Norkin and Levangie, 1983). The TMT joints regulate the position of the metatarsals and phalanges during walking. In the transition from flatfoot to push-off, the lateral metatarsals rotate so that the toes maintain contact with the ground (Scott and Winter, 1993).

Motion at the TMT joints is restricted by the shapes of the bones and the many ligaments that bind them tightly to one another. Sammarco (1989) stated that TMT motion is gliding, although Scott and Winter (1993) considered the joints to be single-degree-of-freedom hinge joints and spoke of TMT flexion. Hicks (1953) found the third ray to be relatively motionless during forefoot inversion and eversion. Ouzounian and Shereff (1989) determined that during dorsiflexion-plantarflexion, the MT3-CF3 joint moved *less* than any of the other TMT joints except for the MT2-CF2 mortise joint. During supination-pronation, the MT3-CF3 joint ranked third out of the five joints in total degrees of motion, behind the MT4-cuboid and MT5-cuboid joints. Scott and Winter (1993) found that there was a decrease in the amount of joint motion from the fifth to the third metatarsal, with the third metatarsal showing only about 2 degrees of movement. Myerson (1989) thinks of the metatarsals as a functional unit.

Joint compression forces were investigated by Manter (1946), who found that the third TMT joint bore *less* compression force than any of the other TMT joints except for the fifth. Cavanagh et al. (1987) found that the midfoot joints bore only about 8% of the total load on the foot during barefoot standing. They also discovered that during peak standing pressures on the foot, the lateral three metatarsal heads, as a unit, bore a load equal to the combined load of the second and first metatarsal heads.

Stokes et al. (1979) studied forces acting on the metatarsal heads during the push-off phase of walking. Not surprisingly, they found that the first metatarsal head bore the most weight, with weight distributions decreasing toward the more lateral metatarsals. In all their measures, forces were highest in the first metatarsal and decreased across the foot to the fifth metatarsal. However, they also stated that MT2 and MT3 may be loaded out of proportion to their sizes when compared with MT1, which may be why MT2 and MT3 are prone to stress fractures in their shafts.

The above discussion indicates that, biomechanically, there seems to be nothing that marks the third TMT joint as being widely

different from the other TMT joints. Other TMT joints exhibit both more and less motion as well as more and less compression than the third TMT joint. If the defect seen in MT3 and CF3 were due to forces across the joint during locomotion and standing, then we would expect to also see similar defects in other TMT joints. In addition, Wilbur (1997) failed to find any demonstrable age or size biases in her large sample, two biases we would expect to see if biomechanics were the cause of the pits. Therefore, it is considered unlikely that biomechanical forces are the cause of the MT3-CF3 pit defect.

Arthritis, infection, and trauma

Degenerative arthritis can often result in morphological changes to the joint surface. For this reason, we considered it in our differential diagnosis. However, it was ultimately excluded for four reasons. First, there appears to be no age bias in the appearance of the defect (Wilbur, 1997), whereas degenerative changes become more common in older age groups. If the pits were caused by degeneration of the articular facet, then they should be more common in older age groups. Second, other signs of degenerative changes on the articular facet (lipping, microporosity, or eburnation) were absent in the area around the pit defect. Tenney (1991) noted a similar lack of arthritic changes at the site of the defect. Third, there is regularity in location of the defect. The pit always involves only the third tarsometatarsal joint, and never the other TMT joints; it is always found in the same part of the articular facet; and it is *always* symmetrical across the joint space, i.e., if it appears on the third cuneiform, it always appears on the third metatarsal. Finally, as we discussed above, biomechanics of the joint also argue against the pits being a result of degeneration. Because there is relatively little movement possible at the third TMT joint, mechanical causes of joint morphology change are unlikely. If the pit were the result of degenerative changes brought about by walking or running, then we would expect to see similar changes in the other TMT joints as well. To the best of our knowledge, this pit defect has never been described in any of the other TMT joints.

Bone infection was excluded from the differential diagnosis for two reasons. First, the bones with the pit defect exhibited no other signs of pathology, such as periostitis, that we would expect with infection. Nor were signs of infection found elsewhere in the associated feet when the other bones were present for examination. Second, the regularity in location and morphology of the pit defect argues against infection as a cause. It seems unlikely that infection would regularly target only the plantar part of the third TMT joint and not also affect other parts of that joint or other TMT joints.

Trauma (fracture or dislocation) was excluded as the cause of the defect because of the regularity in location and expression of the defect and the lack of pathology other than the pit at the affected or nearby joints. Trauma to the third cuneiform is very rare in the clinical literature. Indeed, Mandracchia et al. (1994) gave “A Rare Tarsal Injury” as the subtitle to their article about a CF3 fracture. They went on to say that fractures of the cuneiform bones are usually associated with dislocations at the midtarsal or tarsometatarsal joints. Fractures, including stress fractures, of the third metatarsal *shaft* are fairly frequent (Alepuz et al., 1996; Urteaga and Lynch, 1995), but fractures of the MT3 *base* are not (Urteaga and Lynch, 1995).

Dislocations of the TMT joints can occur. These injuries can be caused by falls from a height, equestrian activities, abnormal twisting of the foot, sports, direct force, and motor vehicle accidents (Faciszewski et al., 1990; Myerson, 1989; Vuori and Aro, 1993). “Subtle” Lisfranc joint injuries have been described, but these affect the first and second TMT joints. No description was found in the clinical literature of a dislocation affecting only the third TMT joint. In addition, Lisfranc joint dislocations result in significant soft-tissue trauma to the TMT joints and often also result in dislocations at the metatarsophalangeal joints (Myerson, 1989). Disruption of the TMT joints caused by dislocation can result in a lack of foot rigidity during the end of the stance phase of walking (Trevino and Kodros, 1995), putting

unnatural stresses on the foot joints. Soft-tissue trauma and biomechanical instability, therefore, could be expected to predispose an individual to degenerative arthritis of foot joints. As we discussed above, degenerative changes were not noted on the bones with pit defects or on surrounding bones where these could be observed. Signs of trauma (healed or unhealed) were also absent from the affected bones. We consider it unlikely that Lisfranc joint dislocation is the cause of the pit defect.

Another type of trauma, osteochondritis (osteochondrosis) dissecans, can cause a localized pit in an articular surface. Osteochondritis dissecans refers to a fracture involving the articular cartilage and the underlying subchondral bone (Lehman and Gregg, 1986), resulting in partial or complete detachment of a fragment of cartilage and bone (Aufderheide and Rodriguez-Martin, 1997). Osteochondral fractures are found most commonly on the medial femoral condyle, the trochlea of the talus, and the capitulum of the humerus (Aufderheide and Rodriguez-Martin, 1997; Pappas, 1981). We do not believe that the MT3-CF3 pits are the result of osteochondral fractures because such fractures usually are found on convex bone surfaces (the third TMT joint surface is relatively flat), they are rare in the midfoot (Lehman and Gregg, 1986), and they do not affect both articulating facets, i.e., they are generally found on only one articular surface in the joint. The pit defect is symmetrical across the joint space.

Tarsal coalition

Tarsal coalition occurs when two adjacent bones fail to separate completely during joint formation (Garn et al., 1976; Gardner et al., 1959). At the close of the embryonic period, the foot usually exists as a homogeneous mass of mesenchyme representing the tarsus, with five tubular projections representing the toes (Gardner et al., 1959; Sarrafian, 1993). Joint formation is initiated in this mass during the early fetal period, when cellular condensations appear in the zones between the future skeletal elements. At the same time, chondrification of the center of each tarsal and metatarsal element is already underway. If joint space

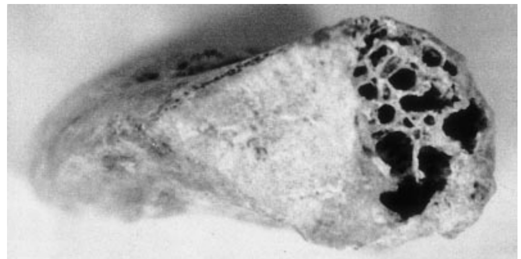

Fig. 3. Large defect on the navicular articular surface of a CF1, reminiscent of that seen on MT3 or CF3.

formation is not completed in time, the two bones may not be fully separated by the time chondrification of the joint area begins. The eventual result is a bridge between adjacent bones that may or may not completely ossify after birth.

We were first led to consider the developmental defect called tarsal coalition as the cause of the MT3-CF3 pits based on five lines of evidence. First, illustrations in Sarrafian (1993, from Pfitzner, 1896) depicted osseous coalition of the third metatarsal and cuneiform, which was shown to occur at the same location as the pit defects. Second, nonosseous tarsal coalition was suggested, based on morphological similarities between the MT3-CF3 pit defect and defects in navicular-first cuneiform joints from archaeological samples (Fig. 3). These navicular-CF1 joint changes were described by Hynes and Romash (1987), who presented a clinical case in which the navicular and CF1 were joined by a "tough synchondrosis of partially calcified cartilaginous material." This synchondrosis was seen radiographically as a joint-surface defect involving both the navicular and the first cuneiform. The limits of the defect were symmetrical across the joint space, and the defect's margins extended into both bones. Hynes and Romash (1987) identified the navicular-CF1 defect as a nonosseous (cartilaginous) tarsal coalition.

Third, we found an archaeological skeleton in which osseous coalition of the MT3-CF3 in one foot was paired with a CF3 containing the pit defect in the other foot (Fig. 4) (Case, 1996). The corresponding MT3 was not recovered. This archaeological skeleton came from the prehistoric Vosberg site (AZ P:13:26 ASU) in central Arizona.

Fig. 4. Tarsal coalition within one individual. **a:** Right CF3, with a large pit defect and postmortem damage above (right MT3 not recovered). **b:** Left CF3 and MT3 from the same individual, fused at the plantar part of the articular surfaces (with normal bones below for comparison). The location of the osseous coalition in the left foot corresponds to the location of the pit defect in the right foot. Photographs by Korri D. Turner.

Direct association of these two defects (osseous coalition and the pit defect) within the same individual strongly suggests that they are related conditions. Fourth, Pfitzner (1896) observed a similar situation in one of his anatomical specimens: osseous coalition of MT3 and CF3 in one foot paired with what he identified as nonosseous coalition (our "pit defect") in the other foot. Although he wrote more than a century ago, Pfitzner remains a frequently cited authority on developmental anomalies of the human foot; he carefully dissected the feet of several hundred cadavers in order to report on the frequency and form of a variety of congenital anomalies, including tarsal coalition. Finally, Pfitzner (1896) illustrated examples of nonosseous coalition of the MT3-CF3 joint surfaces, seen here in Figure 5. This drawing from 100 years ago of Pfitzner's affected anatomical specimen depicts exactly what physical anthropologists have been seeing in archaeologically recovered dry bone. Because Pfitzner did his own dissections, he observed the actual nonosseous bridges between MT3s and CF3s along with the associated bony changes.

Tarsal coalition as the etiology for the pit defect succeeds where the other considered etiologies consistently fail, by encompassing the regularity in both the location and the appearance of the defect. Tarsal coalitions always affect both bones of the joint (explaining the symmetry seen across the joint space); and although there are variances, tarsal coalitions commonly affect the same portions of a joint in different individuals (explaining the observation for MT3 and CF3 that it is only the plantar part of the joint that is affected). Indeed, Trolle (1948) described MT3-CF3 coalition as "in all cases [being] partial only, always on the plantar side, generally extending only over the plantar one-fourth or one-fifth [of the joint],

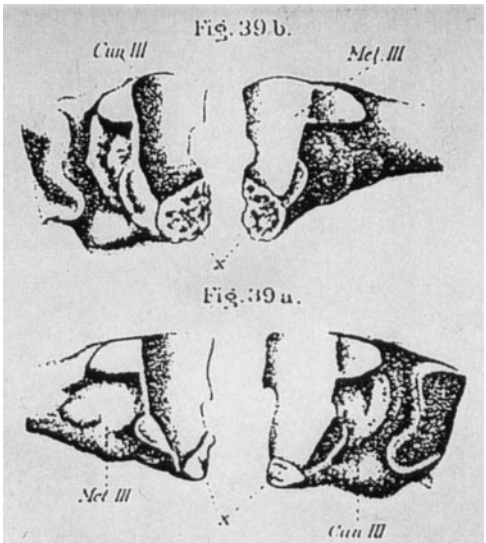

Fig. 5. Nonosseous tarsal coalition from Pfitzner (1896). His illustration from 100 years ago clearly shows the pit defect commonly found in archaeologically recovered skeletal samples.

never more than half." The description by Trolle (1948) echoes that of Pfitzner (1896), who stated that "the fusion never includes more than a third of the contact surfaces... Typically, my observed form of fusion affects the plantar region of the joint" (translated from German).

Three major forms of tarsal coalition have been identified: synostosis (osseous union), synchondrosis (cartilaginous union), and syndesmosis (fibrous union) (Rosen, 1984; Sartoris and Resnick, 1985). A fibrocartilaginous form of coalition has also been identified (Kumai et al., 1998). Radiographically, a cartilaginous union is said to involve joint space narrowing with cortical irregularity, whereas a fibrous union involves only joint space narrowing (Kumar et al., 1992). However, Conway and Cowell (1969) said that if a fibrous or cartilaginous coalition exists, then radiographically the dense cortical surface of the affected bones will be indistinct and irregular at the point of coalition. Cortical irregularity, then, is a feature of nonosseous tarsal coalition.

It has been noted in the clinical literature that some tarsal coalitions may be acquired after birth, i.e., not all are the result of developmental disturbances. Some of the postnatal causes given are inflammation (e.g., rheumatoid arthritis), infection (e.g., syphilis acquired at birth), joint changes secondary to trauma, degenerative joint disease, and other arthropathies (Conway and Cowell, 1969; Pachuda et al., 1990; Sartoris and Resnick, 1985). However, these alternative causes have already been discussed as part of the differential diagnosis and have been found wanting as explanations for the MT3-CF3 pit defect. The following discussion relates only to coalition as a prenatal developmental disturbance.

Coalition between two cartilaginous elements has been observed through dissection in fetuses, demonstrating that the joining of these bones is not always due to trauma, arthritic degeneration, biomechanical forces, or the incorporation of accessory ossicles (Harris, 1955; Trolle, 1948). Trolle (1948) found several examples of synchondroses (cartilaginous coaltions) during examinations of 250 pairs of feet obtained from spontaneous and induced abortions in Copenhagen, Denmark. Most forms of synchondrosis were rare, occurring in fewer than 1% of the fetuses examined. The only common form that Trolle (1948) reported was that between MT3 and CF3, which he found in 6.8% of the 250 pairs of feet.

The occurrence of tarsal coalition is not age-related, unlike its clinical course (pain or other symptoms), which is based on the ossification times of the affected bones. Tarsal coalitions become clinically important in late childhood and adolescence, when the cartilaginous bone precursors are ossifying and activity levels are increasing, resulting in pain due to abnormal biomechanics of the affected joints (Mosier and Asher, 1984; Sartoris and Resnick, 1985). This does not mean that tarsal coalitions are not present before these ages. Rather, the fibrous or cartilaginous nature of the coalition and the cartilaginous nature of the surrounding immature bones mean that enough flexibility is present in the joints that symptoms of pain do not occur at younger ages (Conway and Cowell, 1969; Sartoris and Resnick, 1985; Stormont and Peterson, 1983). Tarsal coalition is frequently found in relatives of individuals who seek treatment for their own

coalition, suggesting a degree of inheritance for many of these conditions. In fact, Leonard (1974) proposed that they are inherited as autosomal dominant traits with "very nearly full penetrance."

Although coalition can occur between any two adjacent bones of the tarsus, it is most frequently reported in the clinical literature between the calcaneus and navicular and between the calcaneus and talus (Stormont and Peterson, 1983). These are medically important coalitions because they are associated with a painful condition called peroneal spastic or rigid flatfoot (Harris and Beath, 1948; Jayakumar and Cowell, 1977). Other varieties of tarsal coalition are less clinically important and hence do not appear in the literature as frequently. Indeed, Conway and Cowell (1969) stated that "tarsal coalition may be completely asymptomatic and present only as an incidental finding." In our own experience with archaeological populations, we have seen osseous and nonosseous coalitions between MT3s and CF3s, tali and calcanei, a talus and navicular, naviculars and first cuneiforms, second and third cuneiforms, a third metatarsal and proximal phalanx, and intermediate and distal phalanges of the toes. It is important to note that not all coalitions progress to osseous fusion. They may remain nonosseous. Pfitzner (1896), who observed MT3-CF3 coalitions in individuals ranging in age from 17–68 years, stated that "bony fusion can fail to appear well into the highest ages." Kumai et al. (1998) likewise found nonosseous coalition in individuals over 60 years of age.

Although not reported in the clinical literature, coalition between the third metatarsal and third cuneiform may be one of the most common forms of tarsal coalition. Trolle (1948), as noted above, found MT3-CF3 coalition in 6.8% of the 250 pairs of feet he examined. Pfitzner (1896, quoted in Sarrafian, 1993), in his classic anatomical study of foot defects, reported that coalitions at the MT3-CF3 joint were by far the most common coalition he encountered. Of the 313 individuals he examined, 10 individuals were affected, suggesting a frequency of approximately 3.2%. Since the sample of Pfitzner (1896) was anatomical rather than clinical, his results should be free of the bias toward dysfunctional conditions inherent in clinical sampling and may provide a more accurate reflection of the frequency of tarsal coalitions in European populations.

CONCLUSIONS

Frequency data for the defect in the common articular surface of the third metatarsal and third cuneiform were presented from several Native American sites in the Southwest and the Plains, as well as data on Japanese and Ainu populations. These data indicate that North American Indians have the highest known frequencies, clustering between 12–20%. European and African populations have lower frequencies. In addition, we documented a range of expression for the defect, from very subtle dimpling or creasing of the articular surface up to very large and deep pits. Statistical comparisons showed that, in general, geographically close samples exhibited similar frequencies.

The etiology of the MT3-CF3 defect was examined, and a developmental defect, tarsal coalition, appears to be the best explanation. Biomechanically induced changes, degenerative arthritis, infection, and trauma were considered as alternate causes. However, each had shortcomings in its explanatory power, mainly that none could completely explain the regularity in location and expression of the defect.

Heiple and Lovejoy (1969) wrote about the antiquity of tarsal coalition, surmising that since it was present in pre-Columbian Native Americans and in European-derived populations, it must be an ancient trait. Our data support their conjecture. The lack of defects in the observed nonhuman primates hints that the defect may be strictly human, although our small sample sizes for the different ape species mean that this point requires further investigation. Our findings concerning the modern Japanese further suggest that the trait occurs in high frequencies not just in North American Indian populations but in populations that are derived from northeast Asia. In addition, the apparent genetic component to tarsal coalition suggests that it may be useful as a skeletal nonmetric trait, as Tenney (1991) first suggested.

Our finding of a range of variation for the defect suggests that the medically based divisions of osseous-cartilaginous-fibrous are perhaps not appropriate for archaeologically derived populations. It is possible that the articular surface dimpling that we observed represents the less restrictive fibrous coalition, while the pit defect represents more restrictive cartilaginous coalition. Because of this uncertainty, we feel that it is inappropriate to use the three classes of clinical characterization. Instead, a simple dichotomy of osseous and nonosseous tarsal coalition is more appropriate to archaeology and paleopathology. Therefore, the pit defect on the third metatarsal and third cuneiform should be described as an example of nonosseous tarsal coalition.

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